cal management and to perform pericardiocentesis as needed. Five million units of penicillin was given daily by vein. An electrocardiogram two hours after the first aspiration of fluid showed lowering of the ST segments (Figure 1). On the fifth hospital day grunting respirations returned and the pericardial rub was no longer audible. On aspiration carried out at that time 30 ml of cloudy, serous fluid was withdrawn. A polyethelene catheter was inserted into the pericardial sac through a No. 13 gauge (French) needle and the sac was irrigated with one million units of aqueous crystalline penicillin. The catheter was left in place and the procedure repeated the following day without return of fluid.

The patient continued to improve although the temperature remained elevated for 10 days. He was maintained on high doses of parenteral penicillin. On the seventeenth hospital day he was discharged completely recovered. Long acting penicillin was given intramuscularly on discharge. During four months of intermittent observation in the outpatient department the child remained well, and an x-ray film showed a moderate decrease in cardiac silhouette.

Comment

Reports in the literature suggest that pneumococcal pericarditis is a result of direct extension from a pneumonic process in the adjacent lung parenchyma.[3,5] It is generally recommended that surgical drainage is the therapy of choice in infants and children.[1,2,4] The patient in the present case was successfully treated with repeated pericardiocentesis and antibiotics alone. Diplococcus pneumoniae, the only organism cultured in the case here reported, is very susceptible to penicillin. However, if a resistant organism is present, surgical drainage would appear to be indicated in order to completely eliminate the offending organism from the pericardial cavity. As was illustrated in the present case, periocardiocentesis can be a useful diagnostic as well as a therapeutic procedure.

Summary

A case of pneumococcal pericarditis in an eight-month-old child is reported. The patient was successfully treated with antibiotics and periocardiocentesis.

REFERENCES

1. Ainger, L.: Acquired Heart Disease in Infants and Children in Brennemann: Practice of Pediatrics, 33; chapter 37, W. F. Prior Co., Hagerston, Md., 1962.
2. Benzing, G., III, and Kaplin, S.: Purulent pericarditis, Amer. J. Dis. Chld., 106:289, 1963.
3. Boyle, J. D., Pearce, M. L., Guzel, L. B.: Purulent pericarditis: Review of literature and report of 11 cases, Med., 40:119, 1961.
4. Crastnopol, P., Kroop, I. G., Levy, G.: Management of pericardial effusions by pericardiectomy, New York J. Med., 582:2367, July 1958.
5. Nadas, A. S., and Levy, J. M.: Pericarditis in children, Amer. J. Cardiol., 7:109, 1961.

Cystic Pulmonary Metastasis Complicating Angiosarcoma Of the Scalp

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ACCORDING TO FREILICH and Coe,[2] a review of the literature from 1918 to 1934 disclosed only 29 reported cases of angiosarcoma, some of which Kinkade[3] believed were not true angiosarcomas. Kinkade reviewed the literature from 1939 to 1949 and "after reading reports of 118 cases labeled with some name suggestive of a malignant vascular tumor," he felt compelled to reject 41 of them "either because there was an inadequate or no histological report, or because . . . the illustrations and texts described a tumor of some other kind." Although he pointed out that the true incidence of angiosarcoma is actually greater than reported, these tumors are not often encountered. The case here reported showed the unusual added features of cystic pulmonary metastasis and hemopneumothorax, demonstrated by x-ray study of the chest and confirmed by autopsy, and adds another possibility to the differential diagnosis of cystic disease of the lung.

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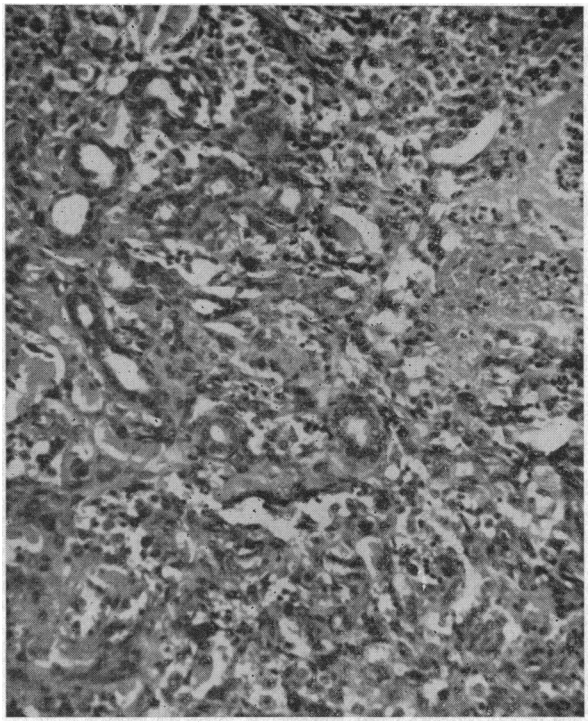

Figure 1.—Photomicrograph of the primary lesion in the scalp—angiosarcoma (×400).

Report of a Case

The patient, a 77-year-old Filipino agricultural laborer, already had two "pimples" on the scalp in April 1960 when he noted a lump on the right side of his head. Soon afterward another lump appeared on the scalp, this time on the left side. When the latter bled in July 1960, the patient consulted a physician. Biopsy (Figure 1) revealed numerous irregular, tortuous and anastomosing vascular channels (often lined by large endothelial cells), and the proliferation of endothelial cells, a few of which had large and deeply stained nuclei. A few mitotic figures were seen. The histologic diagnosis was "hemangioma of the scalp." The patient thereupon was referred to the surgical clinic of the University of California Medical Center at Los Angeles, and on 17 October another biopsy showed tumor cells with large pleomorphic nuclei and abundant cytoplasm scattered throughout the dermis, frequently forming vascular spaces. Several mitotic figures and a few tumor giant cells were present; some cells had spindle shaped nuclei. Many lymphocytes and neutrophils were seen in the dermis, as well as a few foreign body giant cells. The histologic diagnosis was "malignant vascular tumor, probably angiosarcoma." Upon review of the slides from the first biopsy, they were interpreted as suggestive of "angiosarcoma arising in hemangioma."

Physicians in the surgical clinic recommended excision to be followed by irradiation. The patient returned to his home and reported to the outpatient department of the Imperial County Hospital on 31 October 1960, with complaint of discomfort in the chest, hemoptysis and bleeding from the scalp.

On physical examination the patient appeared well developed and well nourished. He was of dark complexion and appeared younger than the stated age of 77 years. The temperature, pulse, respiration and blood pressure were within normal range. On the scalp there were five separate round tumors, each with the following characteristics: Raised indurated borders, soft central umbilication, a black crust with underlying spongy and bloody tissue, and an irregular margin of bluish-red discoloration about 1 cm wide. From the umbilicated center of the tumor above the left ear soft white tissue protruded. The tumor located in front of the left tragus covered the temporo-mandibular area, and an irregular bluish-red discoloration of the skin extended from it to the left eye. The two lesions on the right side of the scalp measured 2.5 cm and 3 cm in diameter, while those on the left were 2, 4, and 6 cm in diameter.

The hemoglobin was 8.2 gm per 100 ml of blood, hematocrit 28 per cent, leukocytes 17,000 per cu mm, with 85 per cent segmented neutrophils, 12 per cent lymphocytes, 2 per cent monocytes, and 1 per cent stabs. Urinalysis was normal and the Venereal Disease Research Laboratory test weakly reactive.

Unexpectedly, x-ray films showed a localized area of pneumothorax at the right costophrenic angle, and the right diaphragm was slightly elevated. A thin-walled cystic annular shadow (2.5 cm in diameter) was in the right apex, and another (3.0 cm in diameter) in the left base. On 10 November 1960 the patient was admitted to the Imperial County Hospital. Skeletal survey showed no abnormalities. Films of the chest now showed seven thin-walled cystic annular shadows in the right lung and two in the left lung. The coccidiodin test was negative, and the prothrombin time 100 per cent of normal.

Radiotherapy was administered and definite regression of the scalp lesions followed. On 29 November 1960 the patient was readmitted to the

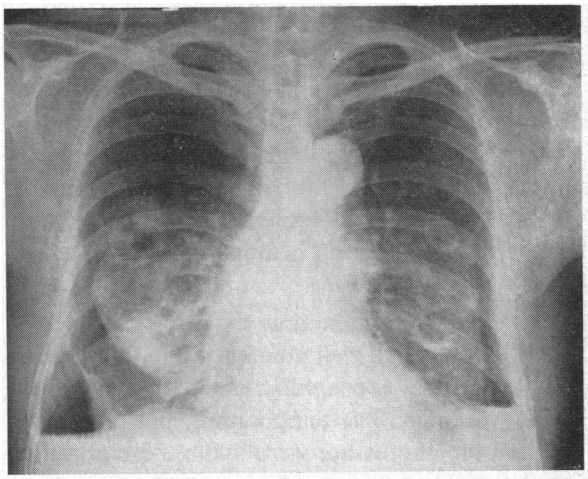

Figure 2.—Posteroanterior roentgenogram of the chest, showing bilateral hydropneumothorax and accentuation of cystic shadows. 29 November 1960.

hospital because of dyspnea and hemoptysis. An x-ray film of the chest (Figure 2) showed bilateral pneumothorax with 40 per cent collapse of each lung. The previously noted cystic shadows appeared accentuated, and at least six cysts were seen in the partially collapsed left lung, the largest being the earliest one detected (October 1960) which now was 4.5 cm in diameter. Pneumothorax was treated by the insertion of large polyethylene tubes into each pleural cavity through the second anterior intercostal spaces, and application of continuous suction. As air and bloody fluid were aspirated, the patient's condition improved dramatically. However, pneumothorax recurred intermittently and the areas of cystic rarefaction appeared to have increased in number bilaterally. A cell block made of the clotted bloody expectoration was reported as negative for tumor cells. The patient's condition became progressively worse, and he died 20 December 1960.

At autopsy multiple lesions consisting of gray-tan granulation-like tissue, surrounded by moth-eaten, mottled, green-brown margins, were seen on the scalp. The right pleural space contained a small amount of bloody fluid and free air. Both lungs were studded with innumerable hemorrhagic cysts that were a few millimeters to 18 millimeters in diameter. The cysts often contained blood or dark brown fluid but were largely air-filled. On microscopic examination of the scalp specimens many, often collapsed, anastomosing channels and capillaries were seen in the dermis and subcutaneous fat. These were lined by atrophic endothelial cells and separated by deposits of hyalinized collagenous connective tissue. A few neoplastic cells possessing irregular, large and hyperchromatic nuclei, and also a few multi-nucleated giant cells were present, as well as many hemosiderin-laden macrophages. In one section, however, the subcutaneous fat was infiltrated by numerous irregular large anastomosing channels lined by anaplastic endothelial cells, which possessed large spindle or irregular and deeply stained nuclei with a scant amount of cytoplasm and long cytoplasmic processes. Many bizarre and giant nuclei were also seen. From multiple sections of the lung it was determined that the cystic lesions were metastatic, since histologic examination of their walls showed the same angiosarcomatous structure as was found in the scalp lesions (Figure 3). In some areas, however, the pulmonary metastases appeared more anaplastic and cellular than the primary focus. The lumina of the cysts often contained blood or partially organized blood clots. Small arteries and veins were invaded by tumor, and "tumor emboli" were seen in the pulmonary vessels.

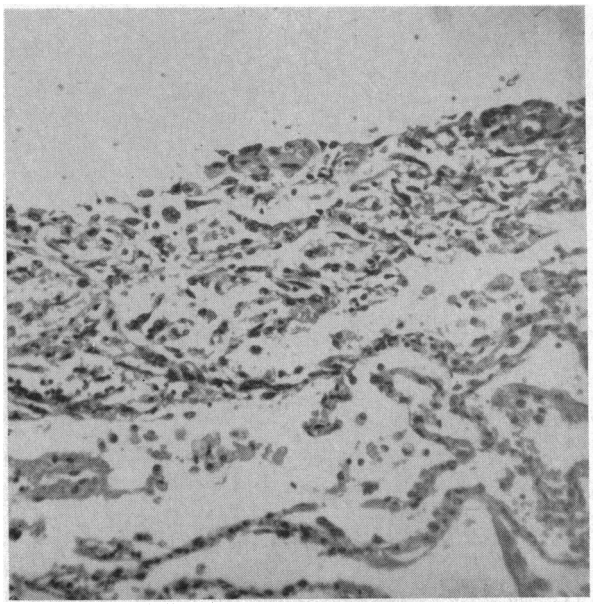

Figure 3.—Photomicrograph showing angiosarcomatous tissue forming the lining and wall of a pulmonary cyst (×400).

The anatomic diagnoses were:

1. Angiosarcoma, extensive, of scalp (previous biopsy excision and radiation therapy).

2. Metastatic angiosarcoma of both lungs with:

a. Tumor embolization of pulmonary vessels.

b. Invasion of blood vessels and pulmonary parenchyma.

c. Hemopneumothorax, right, secondary to rupture of hemorrhagic cystic metastasis.

Discussion

Several instances of angiosarcomatous pulmonary metastases have been reported.* In five of six cases of angiosarcoma of the breast reported by McClanahan and Hogg,[8] pulmonary metastasis developed—"hemoptysis was almost a constant feature late in the disease and usually appeared before the roentgenograms confirmed pulmonary metastases." Their review also summarized nine reported cases of angiosarcoma of the breast. In six, spread to the lung were demonstrated. In the second of two cases of angiosarcoma of the liver described by Bloch[1] there were pulmonary lesions described as numerous, scattered, circular areas up to 0.5 cm in diameter, composed of "a white center surrounded by a hemorrhagic rim. . . . In the lungs the tumor nodules were limited to the thickened pleura and septa." A case of primary angiosarcoma of the pleura with repeated accumulation of bloody pleural effusion was reported by Stout.[13] Lorber[5] described a three-year-old girl with hematuria in whom miliary pulmonary opacities developed and later became coarser. At autopsy "the lungs were studded throughout with a multitude of small hemorrhagic nodules 0.1 to 1.0 cm in diameter." Microscopic sections of the nodules were cavernous and many of the spaces were filled with blood or proliferating endothelial cells. Lane[4] reported the case of a 69-year-old woman with angiosarcoma of the face and scalp, and autopsy revealed "numerous discrete, firm, red spherical nodules (up to 3.0 cm in diameter)" in the substance of both lungs. These nodules revealed "vascular spaces lined by a single layer of endothelium and filled with erythrocytes and blood clot. In parts the spaces roughly conform to the shape of the alveoli by whose walls they are supported. . . . In other places both alveolar walls and the walls of the vascular spaces have broken down, so that there is variation in size and shape of the spaces, and at the periphery the edge is indefinite and blood seeps into the adjacent alveoli." Lane[4] and Tibbs[15] demonstrated tumor tissue in the lumen of pulmonary vessels. Lane concluded that the lesions in the cervical nodes and lung were embolic. In the second of the two cases reported by Bloch[1] in 1958, as well as in the present case, angiosarcomatous tissue was seen invading the walls of pulmonary vessels in the lung. Stout[13] considered spread and infiltration as occurring by "a process similar to the formation of capillaries in granulation tissue" which is manifested by "sprouting of endothelial cells from pre-existing capillaries, forming first a solid cord which secondarily becomes canalized." The frequency of hemotogenous spread was also alluded to by Stout.[13]

In the present case it would have been interesting to study a film of the chest taken before the appearance of the lesions in the scalp, but none had been taken. Nonetheless, it is logical to assume that the pulmonary lesions were secondary deposits arising by embolic spread from the primary process in the scalp, even though the lung lesions showed more pronounced anaplasia and cellularity than the primary site. This disparity has been noted by others.[6,9,11,14] This case satisfies the criteria mentioned by Stout[13] as being essential for the diagnosis of malignant vascular tumors, the constant features of which are: "(1) The formation of atypical endothelial cells in greater number than are required to line the vessels with a simple endothelial membrane, and (2) The formation of vascular tubes with a delicate framework of reticular fibers and a marked tendency of their lumens to anastomose." In addition to these features, actual invasion of the pulmonary vessels was demonstrated as well as embolization of tumor tissue into the vessels of the lungs.

Summary

A case of angiosarcoma of the scalp with the development of unusual cystic metastasis in both lungs has been presented.

Subsequent rupture of these cysts caused bilateral hemopneumothorax as shown by serial x-ray films of the chest.

*Reference Nos. 1, 2, 5, 7, 8, 10, 12, 13, 15.

REFERENCES

1. Bloch, C.: Malignant hemangioendothelioma. Report of two cases and review of literature, J. Mt. Sinai Hosp., N.Y., 25:115-127, March-April 1958.

2. Freilich, E. B., Coe, G. C.: Angiosarcoma: Case report and review of the literature, Am. J. Cancer, 26: 269, February 1936.

3. Kinkade, J. M.: Angiosarcoma: A review of the literature, Ann. Otol. Rhin. & Laryng., 58:159, 1949.

4. Lane, O. G.: Cutaneous angiosarcoma with metastases, Brit. J. Cancer, 6:230-235, 1952.

5. Lorber, J., Ackerley, O. G.: Malignant hemangioendotheliomata simulating miliary tuberculosis and neu-

roblastoma of the adrenal, Proc. Roy. Soc. M. London, 51:288-290, April 1958.

6. Mallory, F. B.: The results of the application of special histological methods to the study of tumors, J. Exp. Med., 10:575, 1908.

7. McCarthy, W. D., Pack, G. T.: Malignant blood vessel tumors—Report of 56 cases of angiosarcoma and Kaposi's sarcoma, Surg. Gynec. & Obst., 91:465, 1950.

8. McClanahan, B. J., Hogg, Lewis, Jr.: Angiosarcoma of the breast, Cancer, 7:586, 1954.

9. Hansom, H. K., Samson, P. C.: Malignant tumors of the greater omentum, Ann. Surg., 100:523, 1934.

10. Raven, R. W., Christie, A. C.: Hemangiosarcoma —A case with lymphatic and hematogenous metastases, Brit. J. Surg. XLI, 169:483-489, March 1954.

11. Robinson, J. M., Castelman, B.: Benign metastasizing hemangioma, Ann. Surg., 104:453.

12. Shore, J. H.: Hemangiosarcoma of the breast, J. Path. & Bact. LXXIV, 2:289, 1957.

13. Stout, A. P.: Hemangioendothelioma—A tumor of blood vessels featuring vascular endothelial cells, Ann. Surg., 118:445-464, 1943.

14. Theile: Virchows Arch, 178:296, 1904.

15. Tibbs, D.: Metastasizing hemangiomata—A case of malignant hemangioendothelioma of the breast, Brit. J. Surgery, 40:465-470.

Progressive Pulmonary Insufficiency Due to Mineral Oil

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CHRONIC aspiration pneumonitis due to lipids has been well studied in the past from a clinical standpoint. However, this entity is occasionally still with us, and particular attention must be paid to distinguishing the advanced form of this disease from other types of chronic obstructive airway disease with similar pulmonary function findings.

Mineral oil pneumonia in humans was first described in the literature by Laughlen[10] in 1925. In the next decade, the articles were chiefly concerned with case reports in children and with experimental work. In 1937, Ikeda[8] reviewed the literature and summarized 106 cases of lipoid pneumonia, more than half of which involved children. In 1943 Sweeney[17] made a further review of 264 cases, and subsequent reports[15] indicated no apparent diminution in incidence. In 1951, Volk and coworkers[18] randomly surveyed 389 chronically ill patients in one institution and found evidence in the sputum or in lung aspirate of lipoid pneumonia in 14.6 per cent of them. These findings resulted in warnings against the use of nose-drops containing oil and the use of such agents was quickly discontinued. There are other sources of oil inhalation, and a residue of people who once used oil nose-drops still exists.

It has been only recently that physiological data have been accumulated in an attempt to delve into the mechanism of the deleterious effects of mineral oil pneumonia. Weill and coworkers,[19] in 1964, reported a 38-year-old service-station attendant with acute lipoid pneumonia from nose-drops who showed significant reduction of lung volume without airway obstruction on pulmonary function testing. Also, arterial oxygen desaturation was present after exercise, along with uneven distribution of inspired air, suggesting a ventilation-perfusion abnormality. A more advanced case of lipoid pneumonia due to mineral oil was reported by Miller and coworkers[13] in a 55-year-old white man with a history of dust exposure and pulmonary disease for at least eight years. The sputum contained fat macrophages, and the patient was partially disabled. Pulmonary function testing in this patient showed obstructive airway disease as evidenced by a decidedly impaired maximum breathing capacity, in addition to restrictive involvement and a more severe ventilation-perfusion disturbance.

The following case is an example of the final result of the progression of mineral oil pneumonia.

Report of a Case

An 86-year-old white man entered La Vina Sanatorium and Hospital Respiratory Disease Unit on 30 June 1964, with complaint of severe shortness of breath and fatigue. He had been well until, three years before admission, he had a bout of pneumonia. Following this, he noticed shortness of breath and a dry cough. This condition responded to treatment and the patient became asymptomatic.

In the year before his admission, he began to have pronounced fatigue and dyspnea, and for eight months before admission these symptoms

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